

# DISSOCIATION WITH INTERFERENCE, RECIPROCAL RHYTHM, AND CORONARY SINUS RHYTHM

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Dissociation with interference and reciprocal beats have many features in common. They have to be differentiated from one another and from auricular parasystole. Herrmann and Ashman (1930) stressed the importance of the auricular rhythm and polarity of the P waves in the bipolar limb leads as differential criteria between the two rhythms, regular auricular rhythm and positive P waves indicating dissociation with interference. Either may occur when the auriculo-ventricular node is an independent pacemaker, or more rarely, when there is a heterogenetic centre of impulse formation in the ventricles. In both orthograde conduction remains intact although it may be impaired. Retrograde conduction is completely blocked in dissociation with interference whereas in reciprocal rhythm it is not altered, although for reciprocal beats to occur it is usually delayed to a certain critical level. The recognition of these disturbances of conduction is important in clinical medicine, for most reported cases have underlying organic heart disease, or are caused by intoxication such as that due to digitalis or to an increased content of pressor substances in the blood. Their presence can only be detected by graphic methods, and on ordinary clinical examination they are likely to be confused with sinus rhythm, or an idioventricular rhythm with extrasystoles in the ordinary sense. Thus either rhythm may be responsible for one variety of pulsus bigeminus.

As early as 1897 Cushny confirmed the existence of two separate foci of stimulus formation in the heart acting simultaneously, the slower rhythm at times interfering with the faster centre of impulse formation: his calculations were made from canine myocardiographic tracings. Later Rothberger and Winterberg (1910), using the electrocardiograph, noted that transient dissociation with interference occurred in the dog's heart during the development of nodal rhythm. In 1923, Mobitz established dissociation with interference as a *pararrhythmia sui generis*. It is characterized by the co-existence of two separate foci of stimulus formation in the heart, usually a slow sinus rhythm and a faster nodal rhythm, the slower rhythm at times interfering with the faster rhythm by conducted beats. These conducted beats form the only link between the two centres, and they occur when a sinus impulse reaches the ventricles outside their refractory period. As in parasystole the slower pacemaker is protected against the faster nodal centre by an entrance block. With regard to reciprocal beats, Decherd and Ruskin (1943), have applied the experimental findings of Schmitt and Erlanger (1928) regarding uni-directional block to explain their return mechanism. They postulate an area of refractoriness in the vicinity of the auriculo-ventricular node, to the neighbourhood of which the impulse returns or sends back a subsidiary impulse to the ventricles. Reciprocal beats are only found in nodal rhythm with pre-ventricular activation.

In clinical practice dissociation with interference is usually observed as a transient phenomenon during the course of changing rhythm, although it may be present over several years (Schott, 1951). Digitalis is an important causative agent, but usually only in the presence of organic heart disease. Schott (1937), however, noticed that small doses of the drug abolished the rhythm in one case. Rheumatic carditis may be the cause of either rhythm. Bain (1939) and Stein and Bartlett (1946) have published cases of dissociation with interference during active rheumatic carditis, and Dock's

case (1928) showing reciprocal rhythm had mitral valve disease. Dissociation with interference is also reported as a feature of coronary insufficiency (Heard and Colwell, 1916; White, 1916; and Burchell, 1949.) Burgess *et al.* (1936), and Espersen and Jorgensen (1947) have reported the par-arrhythmia during the hypertensive crises of phæochromocytoma, presumably due to the increased content of pressor amines in the blood.

#### CASE 1. DISSOCIATION WITH INTERFERENCE

In July, 1951, a man, aged 27 years, was under investigation for symptoms other than cardiovascular. A routine electrocardiogram, however, revealed marked intra-auricular block and complete left bundle branch block (Fig. 1). The lesions in the conducting tissues were considered static and most probably the result of past rheumatic carditis. He was not seen again until May, 1954, when he was admitted to hospital with congestive heart failure. The pulse was very slow and intermittent and on auscultation auricular contractions were clearly audible. A tentative diagnosis of complete auriculo-ventricular dissociation was made compli-

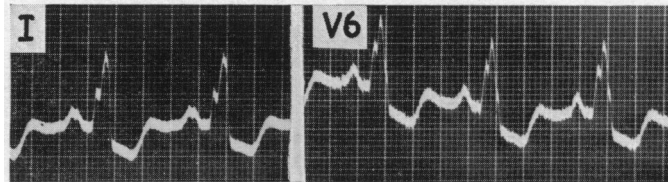


FIG. 1.—Case 1. Intra-auricular block, and left bundle branch block. Time marker 0.04 sec.

cated by 'extrasystoles' in the ordinary sense of the word. Electrocardiography, however, confirmed that the correct diagnosis was dissociation with interference. Subsequently serial electrocardiography was carried out using long strips of lead II to determine the course of changing rhythm. Cuttings from these records are shown in figures of this case, and Table I records the average values of the measurements calculated from the long records taken. There is a marked sinus bradycardia, probably due to 2:1 sino-auricular block, which is

TABLE I  
DISSOCIATION WITH INTERFERENCE.—CASE 1

Fig. No.	1	2	3	4	5	6
Rhythm	Sinus rhythm	Dissociation with interference	Dissociation with interference	Sinus bradycardia	Complete A-V dissociation	Sinus rhythm auricular extrasystoles
P-R	0.2	—	—	0.24		0.18–0.26
Ventricular rate	69.7	48.4	44.4	51	34	44
Auricular rate	69.7	42.8	40	51	66	44
Cycle length	0.86	1.24	1.35	1.16	1.74	1.36
Cycle length after conducted beats	—	1.24	1.35	—	—	—

the true cause of the dissociation with interference. The auricular rate varies between 39 and 77 beats a minute, and the ventricular rate between 33 and 71. Amyl nitrite increased the auricular rate to 48 beats a minute abolishing the conducted beats (Fig. 2) but it had no influence on the ventricular rate. This should be compared with Case 2 (Fig. 10) where this drug caused a marked nodal tachycardia. Dissociation with interference was first noted (Fig. 2) five days after the patient had received 0.5 mg digoxin daily, and it was

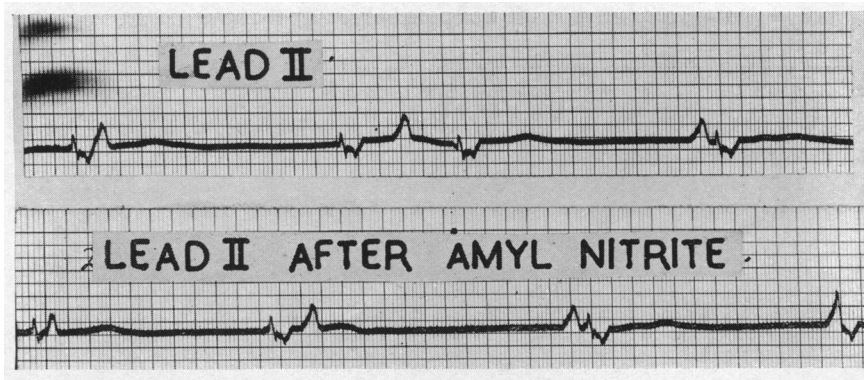


FIG. 2.—Case 1. Effects of amyl nitrite. Dissociation with interference. Upper tracing control. Time marker 0.02 sec.

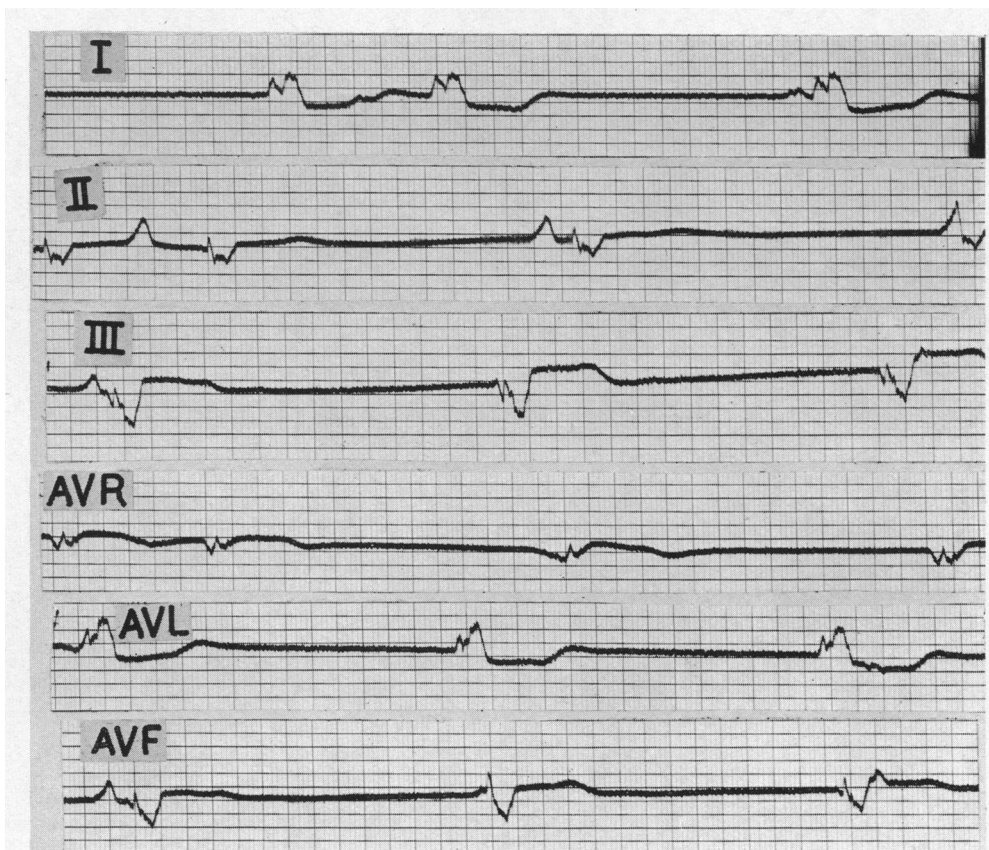


FIG. 3.—Case 1. Dissociation with interference with conducted beats. Time marker 0.02 sec.

found to persist five days after digitalis had been stopped, the ventricular rate having fallen from 48.4–44.4 beats a minute (Fig. 3). No further incidence of the recurrence of the pararrhythmia was recorded in the serial records of this case and it was most likely due to digitalis action. Further cardiograms recorded at intervals of a few days demonstrated sinus bradycardia with first degree heart block (Fig. 4), the P–R interval varying from 0.18–0.26 sec. Fig. 5 shows complete auriculo-ventricular dissociation with a fast sinus rate.

The last tracing (Fig. 6) was recorded when it was noted clinically that the patient's pulse was completely irregular. Multiform auricular extrasystoles with runs of auricular tachycardia (Fig. 6, 6) are seen. Frequently the P waves of the extrasystoles are inverted, the P-R intervals of these beats being 0.26 sec. Occasionally these beats occur in groups (Fig. 6, 5). Scherf and Harris (1946) distinguish such beats as coronary sinus rhythm. The coupling of the multiform extrasystoles varies from 0.92 to 1.16 sec., suggesting that each type arises from a separate auricular focus.

This case, therefore, presents varying rhythms, including dissociation with interference, complete auriculo-ventricular dissociation, and multiform auricular extrasystoles (including coronary sinus beats), which originate in polytopic foci in the auricles.

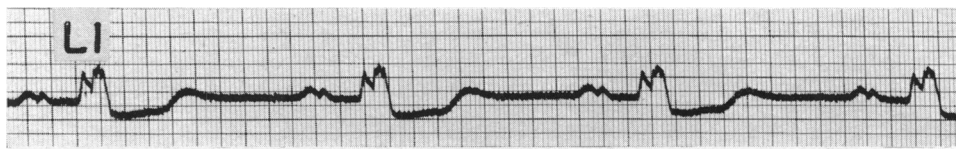


FIG. 4.—Case 1. Sinus bradycardia with first degree A-V block. Time marker 0.02 sec.

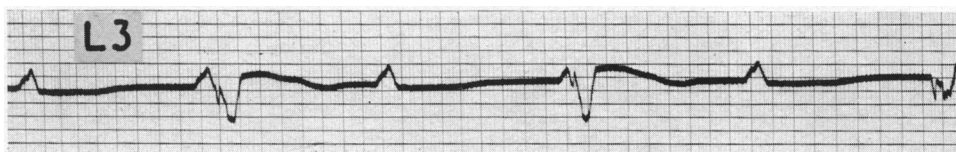


FIG. 5.—Case 1. Complete A-V dissociation. Time marker 0.02 sec.

#### CASE 2. RECIPROCAL RHYTHM

A man, aged 45 years, was noted to have a slow pulse in February, 1954, when he was undergoing investigation for symptoms other than cardiovascular. A routine cardiogram revealed that he had nodal rhythm with pre-ventricular activation (Fig. 7). Subsequently, during July, 1954, serial electrocardiography was carried out as the patient had developed an irregular rhythm due to the occurrence of reciprocal beats. The effects of exercise and of various drugs on the reciprocal beats were studied. Control cardiograms were done under resting conditions before each assessment, and long strips of lead II of 100 complexes or more were used for calculations. Table II represents the average values calculated from the related tracings. Reference

TABLE II  
RECIPROCAL RHYTHM.—CASE 2

Fig. No.	7	8	9			10		11		12		
			A	B	C	A	B	A	B	A	B	C
Ventricular rate	42.8	42.8	35.7	50	40	50	100	40	55.5	30	40	43
P-R	—	—	—	0.16	—	—	—	—	—	0.14	0.16	—
R-P	0.08	0.1	0.18	—	0.06	0.14	0.06	0.06	0.06	—	—	0.14
R-P before reciprocal beats	—	0.2	—	—	—	0.2	—	—	—	—	—	0.32
P-R before reciprocal beats	—	0.3	—	—	—	0.32	—	—	—	—	—	0.32

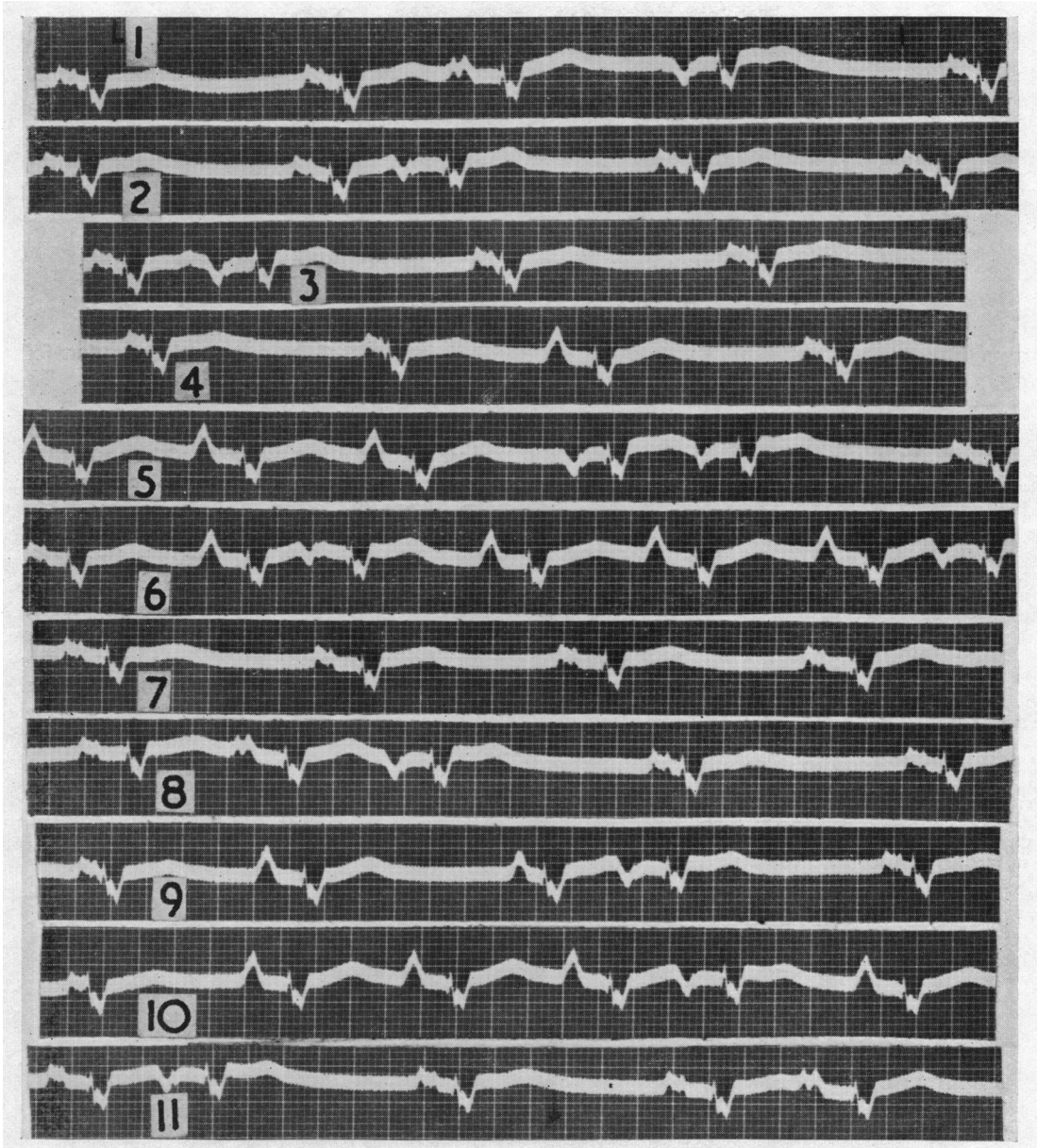


FIG. 6.—Case 1. Multiform auricular extrasystoles. Time marker 0.04 sec.

to the control records (Fig. 7, 8, 9A, 10A, and 11A) reveals that the nodal rate varies spontaneously between 35.7 and 50 beats a minute. They also show variation in the duration of the R-P interval between 0.08 sec. and just under 0.2 sec. Calculations from long tracings of over 250 complexes indicated that reciprocal beats only occurred when the R-P interval was 0.2 sec. or more, so demonstrating the importance of delayed retrograde conduction in the incidence of the re-entry mechanism. It is noted also that the rate of retrograde conduction was independent of the nodal rate under resting conditions. Thus the R-P interval of Fig. 7 is 0.08 sec. with a nodal rate of 42.8 beats a minute, whereas the R-P interval of Fig. 9A is 0.18 sec. with a slower nodal rate, and in Fig. 10A the R-P interval is 0.14 sec. with a slightly faster nodal rate. There is an

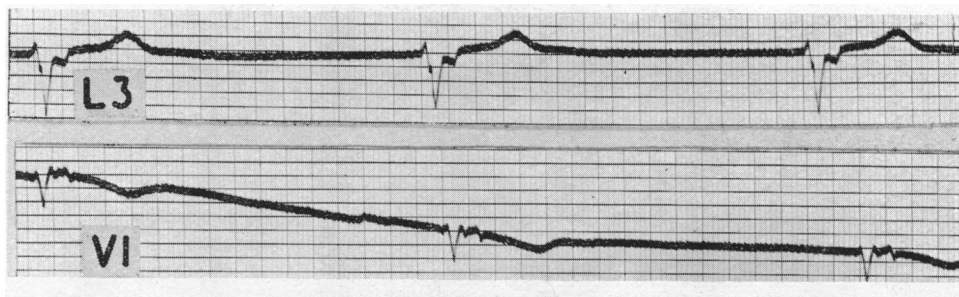


FIG. 7.—Case 2. Nodal rhythm. Time marker 0.02 sec.

inverse relationship between the R-P intervals and the P-R intervals of the reciprocal beats that follow them. In these tracings the R-P intervals are shorter than the P-R interval of the reciprocal beats. This is unusual, as it indicates that retrograde conduction takes place more readily than orthograde conduction (Gravier *et al.*, 1939; Decherd and Ruskin, 1943; Blumgart and Cargill, 1930). Fig. 12A is unique in that it shows sinus rhythm with marked bradycardia, which is most probably the true cause of the nodal rhythm. The P waves are abnormal, due to intra-auricular block. Attention is also drawn to the configuration of the complexes of lead V1 in Fig. 7 and 8. A small secondary r wave in Fig. 7 is suggestive of delayed activation of the right ventricle. The reciprocal beats of lead V1 in Fig. 8 conform to those of a complete lesion of the right bundle, and thus they reveal a latent right bundle branch block. The ventricular complexes of the reciprocal beats in all the records vary in shape due to varying degrees of ventricular aberration.

The effects of strenuous exercise are recorded in Fig. 9. The control tracing has no reciprocal beats as in no incident did the R-P interval exceed 0.2 sec. Fig. 9B and C are cuttings from 100 ventricular complexes recorded immediately following strenuous exertion. The first ten complexes (Fig. 9B) demonstrate a change in the type of nodal rhythm to that with pre-auricular activation and a normal P-R association and increased nodal rate (coronary sinus rhythm, as in Fig. 6). The following complexes (Fig. 9C) confirm a reversal to the original type of nodal rhythm but with marked shortening of the R-P interval to 0.06 sec., the heart rate having returned almost to its original level. Thus exercise resulted not only in an increased nodal rate, but also speeded up retrograde conduction. These are two separate effects not dependent on one another as shown above and probably result from an increased sympathetic tone. Exercise, therefore, abolishes the reciprocal rhythm by shortening the R-P intervals. It was also noted that auricular extrasystoles occurred after exercise (Fig. 9D).

The inhalation of amyl nitrite resulted in nodal tachycardia (Fig. 10B). The nodal rate was doubled but the type of nodal rhythm was unchanged. There was shortening of the R-P intervals from 0.14 sec. to 0.06 sec. and the reciprocal beats were abolished. Before the development of nodal tachycardia, however, there was an intermediate phase of complete irregularity of the heart due to a marked increase in the number of reciprocal beats (Fig. 10B, 1 and 2), the R-P intervals in such cases remaining at 0.2 sec. Amyl nitrite therefore has two separate effects. Increased nodal rate occurs immediately and is initially associated with increased reciprocation, due to the fact that in the transient period retrograde conduction is not altered in rate, and the increased incidence of the ventricular responses increases the incidence of the reciprocal beats. In a few seconds, however, retrograde conduction is speeded up and the nodal tachycardia becomes regular with very much shortened R-P intervals.

Reports of the effects of atropine on reciprocal rhythm differ. Dock (1928) noted an increase in the incidence of reciprocal beats with prolonged R-P intervals. Bishop (1921) found atropine a specific remedy for the relief of one of his patients suffering from palpitations due to reciprocal beats. In our case we were unable to precipitate reciprocal beats by giving 1/50 grain atropine. Fig. 11B, taken after this dose records an increase in the nodal rate of 55 beats a minute from 40 beats a minute in the control. The R-P intervals remained unaltered. It may be noted that the R-P interval of the control cardiogram (Fig. 11A) is the shortest of the series (0.06 sec.), and therefore the atropine had the opportunity to reveal any effect it may have had on retrograde conduction.

The control cardiogram (Fig. 12A) before the administration of 0.25 g. quinidine demonstrates sinus rhythm with marked bradycardia and normal P-R association. Serial records were taken one hour after quinidine (Fig. 12B), and then one hour later (Fig. 12C). Quinidine suppressed the sinus pacemaker and



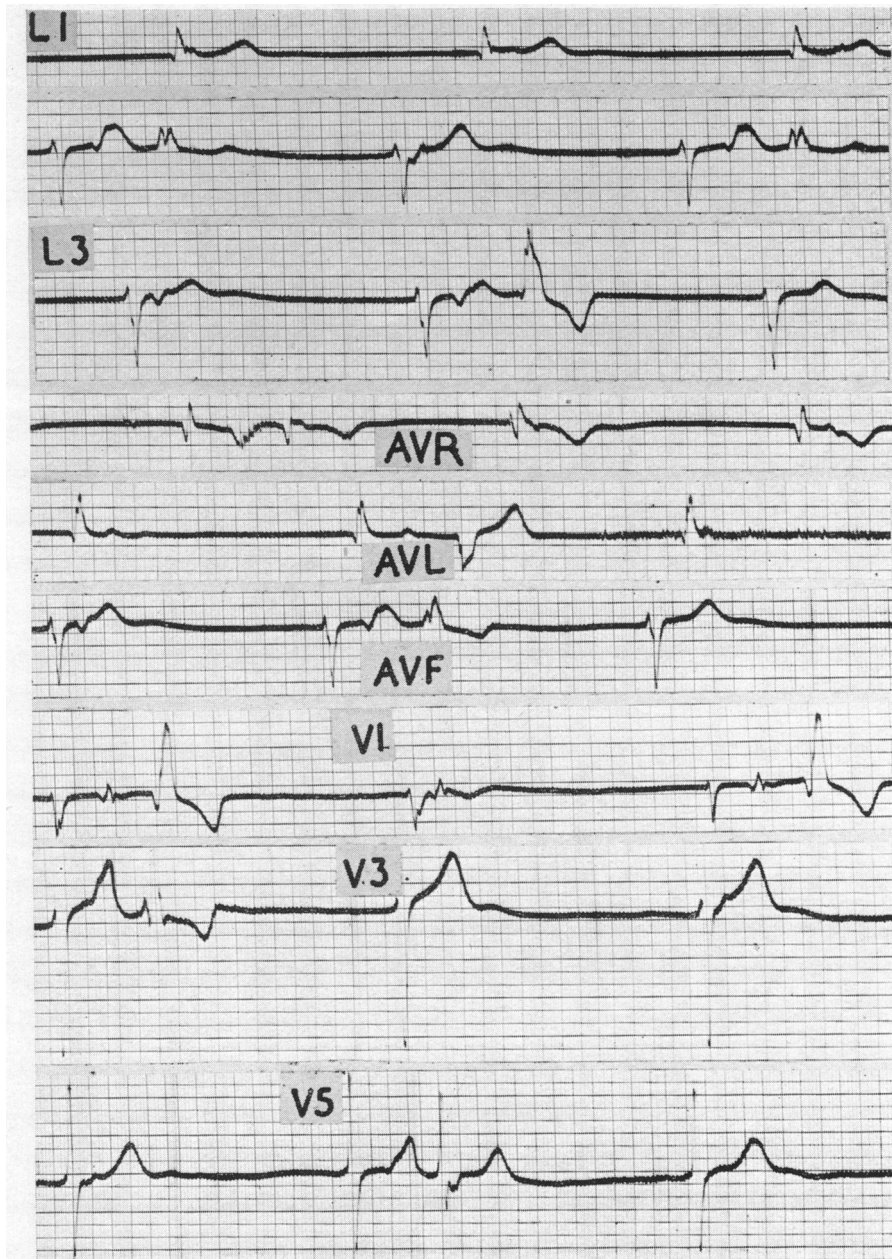


FIG. 8.—Case 2. Nodal rhythm with reciprocal beats. Time marker 0.02 sec.

incidentally increased the heart rate by transferring the pacemaker to the auriculo-ventricular node. Nodal rhythm with pre-auricular activation is recorded in Fig. 12B, and of course, no reciprocal beats. The P-R association is normal which raises the question of coronary sinus rhythm (see Case 1, Fig. 6). An hour later pre-ventricular activation with R-P intervals of 0.14 sec. occurred (Fig. 12C). Twenty reciprocal beats were recorded within 100 ventricular complexes, and the preceding R-P intervals were 0.32 sec., which are the longest of any of the series recorded. Quinidine therefore suppressed the sinus rhythm in this case causing a reversal to nodal rhythm. At first there was pre-auricular activation but after two hours,

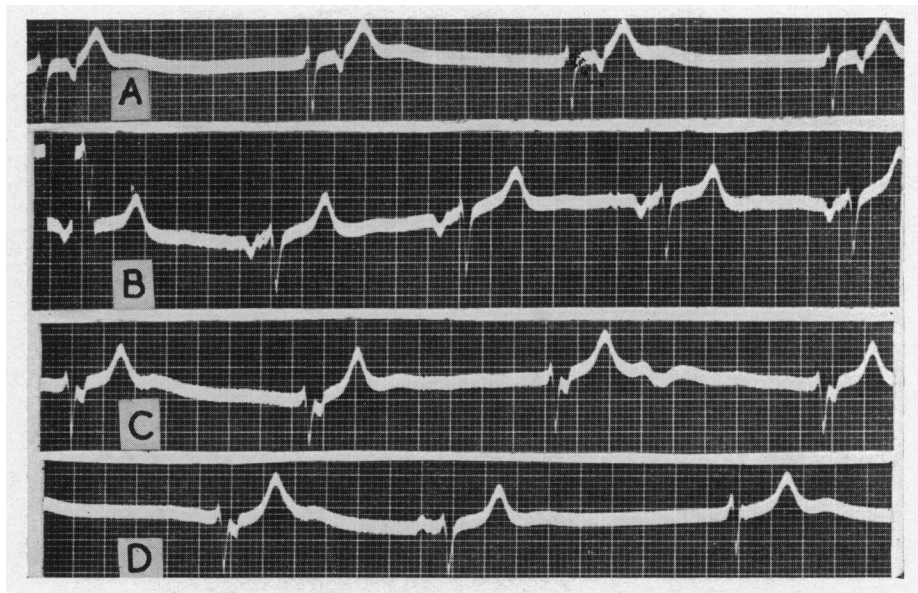


FIG. 9.—Case 2. Effects of exercise. (A) Resting control. (B) Immediately after exercise. (C) Shortly after exercise. (D) Auricular extrasystole. Time marker 0.04 sec.

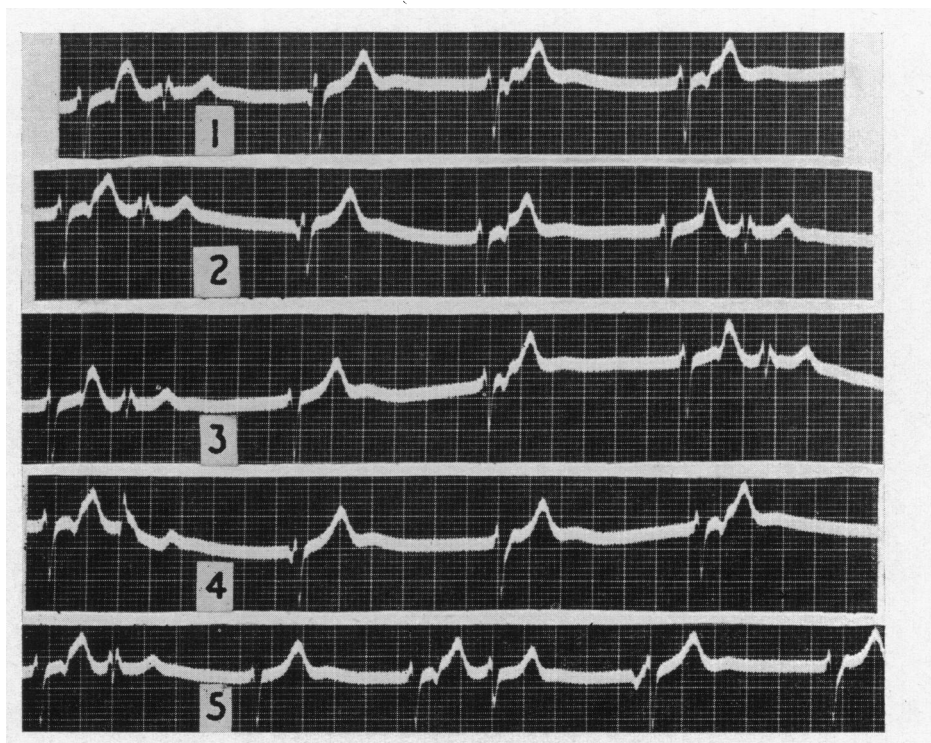


FIG. 10A.—Case 2. Control before amyl nitrite. Nodal rhythm with reciprocal beats. Time marker 0.04 sec.



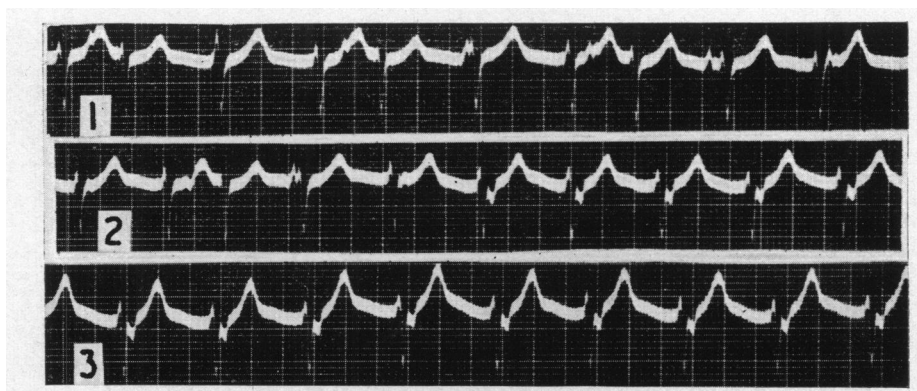


FIG. 10B.—Case 2. Effects of amyl nitrite on reciprocal beats. Time marker 0.04 sec.

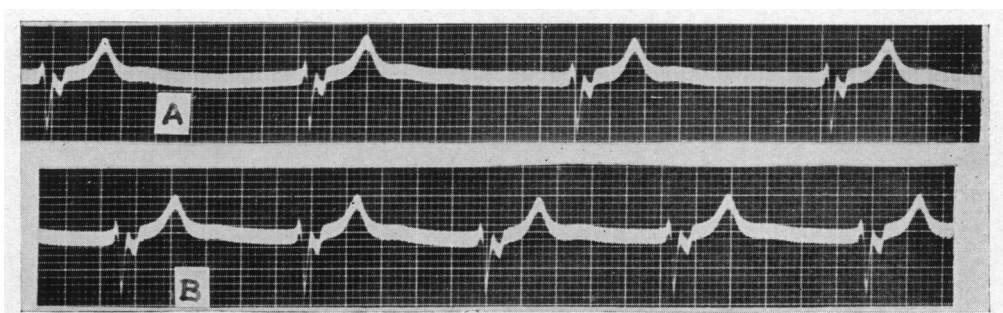


FIG. 11.—Case 2. Effects of atropine. (A) Control. (B) After grain 1/50 atropine. Time marker 0.04 sec.

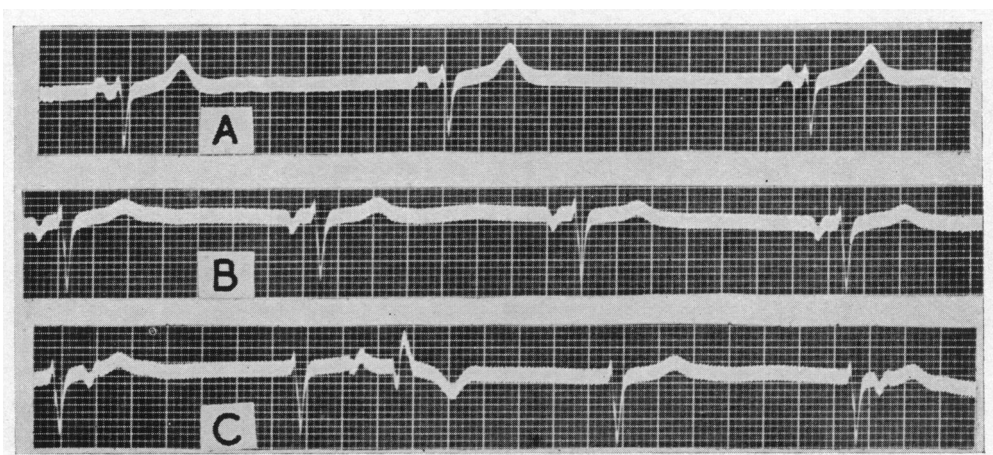


FIG. 12.—Case 2. Effects of quinidine. (A) Control. (B) One hour after quinidine. (C) Two hours after quinidine. Time marker 0.04 sec.

pre-ventricular activation occurred with prolonged R-P intervals and an increase in the incidence of reciprocal beats. Thus retrograde conduction was delayed by quinidine, but in this case there was no evidence of effect of the drug on orthograde conduction of the reciprocal beats themselves. The P-R interval of these beats (0.3–0.32 sec.) was little altered from that of the control record (Table II).

Certain additional features in these records are of basic physiological interest. Inspection of the complexes following the reciprocal beats reveals a change in the sequence of auricular and ventricular activation when compared with the complexes of the dominant rhythm. Scherf and Shookhoff (1925) demonstrated that the temporary change in the position of the P waves in the beats following the extrasystoles is dependent on the speed of conduction in the ventricles. They showed that ventricular extrasystoles which were not conducted backwards to the auricles (as reciprocal beats cannot be) were followed by complexes with pre-auricular activation in the type of nodal rhythm in this case. This apparent "shift of the pacemaker" is due to fatigue of the conducting pathways in the ventricles as the result of the extra beat, so that when the next nodal beat occurs, the auricles are found more responsive than the ventricles and pre-auricular excitation results.

The area close to the orifice of the coronary sinus has been shown to possess a high degree of automaticity (Scherf, 1944). Such coronary sinus beats have a normal P-R interval in contrast to nodal rhythm with pre-auricular activation in which the P-R intervals are greatly shortened. The P-R intervals of the extrasystoles with inverted P waves in Case 1 (Fig. 6) are 0.28 sec., and in Case 2 (Fig. 9B and 12B) the P-R intervals are 0.16 sec. (Table II). These records may therefore be interpreted as coronary sinus rhythm. However, the distinction from nodal rhythm cannot be made with certainty as disturbances of conduction may be present and these would profoundly affect the P-R association.

Clinically both cases had similar symptoms of severe upper abdominal pain attributed to pylorospasm. They were believed to be suffering from a duodenal ulcer, but special investigations failed to prove this diagnosis. However, the symptoms were severe enough to warrant laparotomy and in neither case was peptic ulceration found. Case 1 had a vagotomy which completely relieved his discomfort. The autonomic nervous system plays an important part in the production of these arrhythmias, and it is possible that a common nervous factor was responsible for the symptoms related to the gastro-intestinal tract.

#### SUMMARY

Serial electrocardiographic records of two cases showing dissociation with interference, reciprocal beats, and coronary sinus rhythm are analysed. The effects of exercise and various drugs on the reciprocal beats in one case are noted. The possible correlation between the patients' symptoms and cardiac conditions are mentioned. Brief reference to some reported cases of these rare rhythms is made.

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